



Milk Fever

With the average milk yield for the NZ national herd steadily increasing in recent years the demands of lactation for the dairy cow to maintain blood calcium concentrations around calving have increased. Unfortunately there is no clear data on the prevalence of clinical hypocalcemia (milk fever) in the NZ dairy herd but it is most likely very variable, especially between regions. The target in seasonal calving herds is <2% of the herd having clinical milk fever.

Incidences of milk fever tend to vary with age and breed. Jersey cows, for example, are more susceptible to milk fever and twice as likely to suffer from it than Friesians.

The reason for this seems to be associated with a decreased capacity to mobilise calcium from bone and possibly a decreased number of Vitamin D3 metabolite receptors in the small intestine. However when other factors are controlled the difference between breeds is likely to be small.

The management and nutrition of the cow during the dry period can have a strong influence on the susceptibility of individual animals to the condition.

The word 'fever' is a misnomer, as body temperature during the disease is usually below normal. Hence a better description of the condition is "clinical hypocalcemia".

CLINICAL SIGNS OF MILK FEVER

The clinical signs of milk fever include:

- Cows that are dull and lethargic
- Ears are cold to the touch and nose is often dry
- Stiffness in the legs

In severe cases:

- The cow becomes recumbent (down)
- Has a kink in her neck ("S" bend)
- The rumen becomes static
- Faeces tend to bulge in the rectum
- Death within 6-12 hours

These well-recognised signs are due to the rapid decrease in calcium concentrations in the blood

which occurs close to parturition (calving). The basic reason for this hypocalcaemia is the inability of some cows to match their rapidly increasing requirements for calcium for milk secretion by absorbing sufficient calcium from their gut, or by mobilising calcium from their own skeleton.

Milk fever depresses rumen contractility, and other disorders such as retained placenta, metritis, dystocia, displaced abomasums and ketosis can often be associated with it. Due to these periparturient disorders and the effect on the rumen, feed intake can drop and worsen the energy status of the already affected cow. This negative energy balance in the postpartum cow can have a strong influence on subsequent fertility.

RISK FACTORS FOR MILK FEVER

- High phosphorus intakes pre calving (i.e. PKE, Broll, DDG in quantities over 2 kgDM/cow/day in the last 2-3 weeks pre calving) as this will reduce the effectiveness of vitamin D in helping the cow absorbing calcium
- High potassium intakes pre calving (i.e. effluent paddocks to be avoided)
- Inadequate amount/length of magnesium supplementation pre calving
- Genetics; Jerseys are two times more likely to get MF than Friesians (Harris 1981; Lean et al 2006)
- Age; older cows are more at risk than younger cows. The risk of milk fever increases with 9% per lactation
- Cows that are either too fat (BCS 6+) or too thin (BCS 3.5 or <) are more likely to get milk fever.
- High DCAD of the pre calving diet; in other words the level of positively charged sodium and potassium ions in the diet is much larger than the negatively charged ions like sulphur and chloride.



PREVENTION OF MILK FEVER

Conventional methods for the prevention of milk fever include:

Method 1:

Magnesium supplementation at 15 to 20g/day at least three weeks pre calving, and throughout early to mid lactation.

Most commonly used sources of magnesium are magnesium sulphate, magnesium chloride (both are anionic types of magnesium and could be used either through water system or mixed in with feed) and magnesium oxide (dusting grass/mixed in with silages) Ideally this needs to be given with a source of easily digestible carbohydrates to encourage uptake.

Method 2:

Lowering the DCAD of the pre calving diet.

This can be done by lowering potassium intake (restricting fresh grass intakes) and/or using anionic salts of magnesium

Method 3:

Restriction of calcium in the pre calving period.

This activates the calcium homeostatic mechanisms. This method is effective only if sufficiently low dietary calcium levels are fed (20g/day). However this might be in

reality very hard to accomplish. Recommendation is to control calcium concentrations in the diet to around 0.4-0.6% before calving.



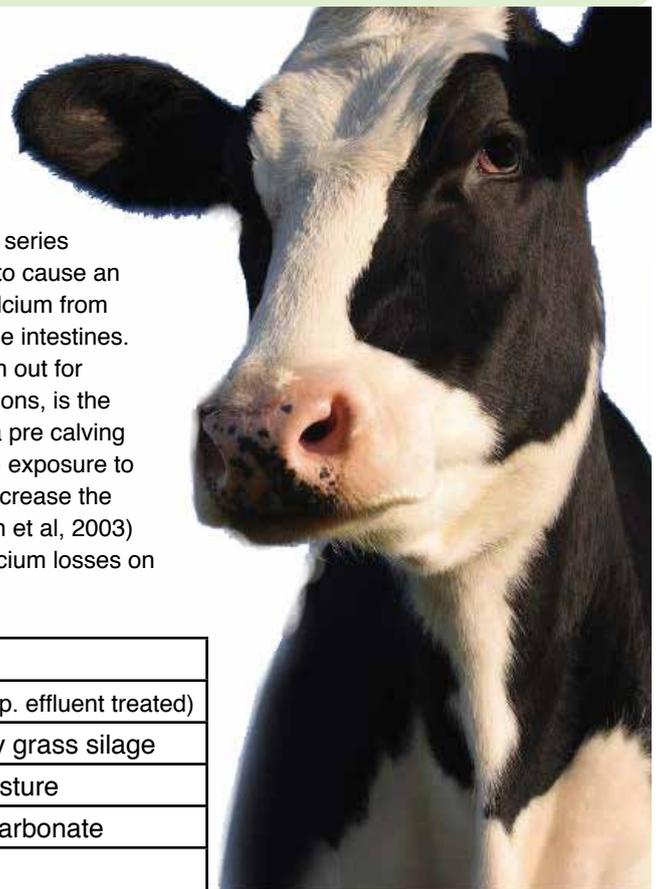
On top of the three methods mentioned above it is important to note that continuation of magnesium supplementation AND the start of calcium supplementation post calving is important to prevent milk fever in the freshly post calving period.

THE DIETARY CATION-ANION DIFFERENCE

It has been shown that pre calving dietary cation-anion difference (DCAD) is strongly correlated to the clinical incidence of milk fever. Reducing DCAD rather than the calcium content of the prepartum ration is now considered the method of choice for preventing milk fever.

DCAD is defined as: $DCAD (mEq/Kg DM) = (Na^+ + K^+) - (Cl^- + S^{2-})$, in other words the difference between positive and negative charged ions in the diet. Because they impact on milk fever independent of DCAD, calcium, magnesium and phosphorus should NOT be included in DCAD equations.

A negative DCAD ration causes mild metabolic acidosis in the blood, which increases the production of Vitamin D3. This has an effect through a series of hormones and pathways to cause an increased mobilisation of calcium from the bone and uptake from the intestines. One important thing to watch out for when feeding low DCAD rations, is the length of exposure to such a pre calving diet as longer (i.e. >3 weeks) exposure to low DCAD pre calving will increase the incidence of milk fever (Lean et al, 2003) due to increased urinary calcium losses on these type of diets.



Low	Moderate	High
Molasses	Maize/whole crop silage	Pasture (esp. effluent treated)
Grains	Cereal hay	High quality grass silage
Most grain based by products	Whole cotton seed	Legume pasture
Protein meals		Sodium bicarbonate
Brewers grains		

For more information contact your local XLVets practice:



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